Fairhaven Board of Selectmen

Meeting Minutes
December 16, 2019

Present: Chairman Charles Murphy, Vice Chairman Robert Espindola, Clerk Daniel Freitas and Town Administrator Mark Rees and Administrative Assistant Vicki Paquette.

The meeting was videotaped on Cable Access.

Chairman Murphy called the meeting to order in the Town Hall Banquet Room at 6:33 p.m.

Mr. Murphy held a moment of silence for Fairhaven Shellfish Deputy Richie Dube who passed away on November 19, 2019. Harbormaster Tim Cox told the Board Mr. Dube was a great man and friend and will be missed. The Board offered condolences to the family of Mr. Dube.

APPROVAL OF MINUTES

Mr. Espindola made a motion to approve the minutes of December 2, 2019 Open Session. Mr. Freitas seconded. Vote was unanimous. (3-0)

TOWN ADMINISTRATORS REPORT

Mr. Rees updated the Board:

Mr. Rees told the Board he has been busy meeting with Department Heads regarding their Capital requests for FY21.

Capital Planning Committee has also started meeting with Department Heads to discuss their capital budget requests and has prioritized them based on factors such as grant funding availability, whether or not is a mandate, how it improves services etc. The next step will be to develop a five year funding plan based on the prioritization.

Mr. Rees said the Fire Chief and the Police Chief met with the Finance Committee to discuss the Public Safety Facility project.

Mr. Rees said he has received notice that Fire Chief Tim Francis has announced his retirement date of January 2021.

Mr. Rees said crews are still busy working to replace and repair sewer lines at the Fire/Police Station. The work on the Assessor's ceiling will continue for a few more weeks as wiring needs to be updated before the project can be completed. These are both town meeting funded projects.

Mr. Rees passed a memo to the Board regarding the EPA Superfund dredging in the Harbor and a study done by Boston University citing the health risks. (Attachment A)

Mr. Freitas and Mr. Espindola expressed they would like to see the EPA send the reports of the monitoring they will be doing regarding the air quality.

COMMITTEE LIASON REPORTS

Mr. Espindola said he had a SRPEDD meeting regarding the route 6 corridor study where they presented two options for route 6. Mr. Espindola read a memo form Mattapoisett resident Bonne DeSousa expressing her concerns. (Attachment B)

Mr. Espindola said the Broadband Study Committee is still working on the Feasibility study.

Mr. Espindola said at the last Marine Resources Committee meeting the members requested advertising for a member to fill a vacant spot. Mr. Rees will advertise in the paper and on the webpage.

Mr. Freitas said he attended the Historical Commission meeting where they discussed the proposed handicap renovations to Town Hall. The Historical Commission expressed their concerns over changes to the historic building and would like to be fully involved with any projects that will take place. Mr. Rees said he will be putting a working group together for the Town Hall renovations that will consist of a member of the Historic Commission and a member of the Commission on Disability and other officials.

Mr. Murphy said that the Commission on Disability will appoint a member to the working group at their next meeting.

Mr. Murphy and Mr. Rees met with Gerry Rooney from the Sister City Committee to discuss updating the lease on the Whitfield-Manjiro house.

SUSTAINABILITY COMMITTEE

Mr. Murphy read a letter from resident Leon Correy who is interested in joining the Sustainably Committee. Mr. Espindola made a motion to appoint Leon Correy to the Sustainability Committee. Mr. Freitas seconded. Vote was unanimous. (3-0)

ABC WASTE DISPOSAL AGREEMENT

Mr. Rees said there has been agreement reached with ABC Disposal, Inc. regarding the Waste Disposal at SEMASS. This agreement allows ABC to use the Fairhaven tonnage allotment at SEMASS and to pay a higher premium above the SEMASS rate. Mr. Espindola made a motion to approve the contract for ABC Disposal, Inc. Waste Disposal Services Agreement. Mr. Freitas seconded. Vote was unanimous. (3-0)

HEDGE STREET PHASE I CHANGE ORDER

Mr. Rees said there is a minor change with the work order due to the project coming in under budget. This will allow for the extra funds to be used for new street signs to complete the job. Mr. Espindola made a motion to approve Change Order #2 to the Town's contract with P.A. Landers, increase the total contract value to $661,923.02. Mr. Freitas seconded. Vote was unanimous. (3-0)

2020 LICENSE RENEWALS

Mr. Rees presented the Board with an updated listing of the license renewals and explained the building commissioner and Fire Department are still inspecting at this time. (Attachment C)

Mr. Espindola made a motion to approve the 2020 liquor licenses that have been inspected by the Building Commissioner. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve the 2020 liquor licenses that are not inspected contingent upon positive review by the Fire Department and Building Inspector. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve all 2020 common victualler licenses. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve all 2020 Car Dealer licenses. Mr. Freitas seconded. Vote unanimous. (3-0)

Mr. Espindola made a motion to approve the 2020 Repair licenses that have been inspected by the Fire Department and the Building Commissioner. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve the 2020 Repair Licenses that are not inspected contingent upon positive review by the Fire Department and Building Inspector. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve all 2020 Lodging House licenses. Mr. Freitas seconded. Vote was unanimous. (3-0)

Mr. Espindola made a motion to approve the 2020 Private Livery License. Mr. Freitas seconded. Vote was unanimous. (3-0)

WEST ISLAND CAUSEWAY LAND

Chairman Murphy read a letter from West Island Improvement Association President Lisa Esten expressing their disappointment over the Board's recent decision regarding the tax title land on the north side of Causeway Road owned by Dan Ristuccia. (Attachment D) Town Counsel Tom Crotty explained that this was a case that was started several years back when the Town was in the process of trying to find a land swap and upon looking into the property on the north side of Causeway Road it was discovered there was a title issue. The land is currently listed on the Assessor's map as one parcel and would need to be subdivided but since the Town no longer needs that land for a swap there is no need to have it certified. The decision of the Board of

Selectmen was to let Mr. Ristuccia clear the title on his own. The Board does not want the Town to expend the money to clear the title.

Mr. Ristuccia's Attorney, Matt Thomas, told the Board he feels the Town should spend the money on clearing the title and foreclose on the property. Mr. Risstuccia would like to take the land back and donate it to the West Island Improvement Association (WIIA). By doing so this will give the Town a clear title on the south side of the Causeway Road.

Ms. Esten feels the Board is not addressing the problem and ignoring the issue will not solve it. Selectman Freitas asked what the intention of the WIIA if they were to acquire the land. WIIA member Dave Hickox explained that the Association would like to make it a private beach for their members and friends of WIIA members. Mr. Freitas would like to see an agreement reached so that in the future there is no misunderstanding about the intentions of the WIIA and the north side. Selectmen Espindola has some concerns over the costs associated with clearing the title and if there is enough money in the FY20 budget. Attorney Crotty is unclear what the costs would be but suggested the Town not spend the money and let Mr. Risstuccia or the WIAA pay for it if they would like to have the property. The Board had concerns about the land being restricted to only WIIA members and not open to the public as it has been for many years and how this would be enforced. Ms. Esten explained that anyone can join for a fee but instead they could just use the town beach and not the private beach. The Board was not comfortable making a decision on this issue but instructed Mr. Rees to put together a working group to discuss this with all the parties involved with a member of the Board, Mr. Ristuccia and his attorney and representative from WIIA. Mr. Espindola made a motion to appoint Mr. Murphy to be the representative of the Board of Selectmen on the working group regarding the land on Causeway Road. Mr. Freitas seconded. Vote was unanimous. (3-0)

DOG PARK LOCATION PRESENTATION

Planning and Economic Director Paul Foley presented two possible locations for a dog park to the Board, the old playground behind Anthony Haven on North Street and the South side of the playground at the former Rogers School. Mr. Foley told the Board what criteria the Dog Park Study Committee and town staff used to determine the two locations. Dog Park Study Committee member Ms. Sallie Lou Johnson had some concerns about the North Street location having restrictions citing that there is a monument in the Park placed there by Mr. Taber that says " for use of school children." Mr. Rees told the Board there is still a lot of work that has to be done before a final decision can be made and because the Board of Public Works are the Park Commissioners, this would have to be brought before their board as well as the Selectboard.

DOG NUISANCE/DANGEROUS HEARING PROCESS

Mr. Rees told the Board there has been a request for Dog hearing and under recent changes in state law, the authority to hear the matter could be "the selectmen of (the) town…… the chief…. Of (the town's) police department, the chief's …. Designee or the person charged with the responsibility of handling dog complaints in a town" (MGL Chapter 140, Section 136A). Mr. Espindola made a motion to appoint the Police Chief or his designee to be the hearing officer as protocol of State Law. Mr. Freitas seconded. Vote was unanimous. (3-0) (Attachment E)

NOTES AND ANNOUNCEMENTS

Mr. Murphy stated the next meeting will be Monday, January 13, 2020

Mr. Espindola said he registered for a training session in Hopkinton regarding "Complete Streets" but due to the storm it was postponed to January 9, 2020 so Mr. Espindola will be unable to make it due to a prior commitment.

Mr. Espindola said he attended the Old-time Holiday this weekend and it was very nice and well attended.

Mr. Freitas said he would like to think about having a vehicle use policy in place for 2020 now that we may be getting some electric vehicles with the grant.

The Board all wished everyone a Happy Holiday season!

At 8:22 p.m. Espindola made a motion to adjourn to executive session and not to reconvene to open session to discuss:

1. Real Estate Matter: Union Wharf Issues, MGL Chapter 30A, Section 21(a) 6
2. Real Estate Matter: North Street/ Lee Miguel Issues, MGL Chapter 30A, Section 21(a) 6

Mr. Freitas seconded. Vote was unanimous. (3-0)

Roll Call vote: Mr. Murphy in favor, Mr. Espindola in favor, Mr. Freitas in favor.

Respectfully submitted,

Vicki L. Paquette

Vicki Paquette
(Approved 01/29/2020)
Attachments:
A: Revised response to BU air paper
B: SRPEDD letter from resident
C: 2020 license renewals
D: Email from Lisa Esten
E: MGL Chapter 140, Section 136A

Gmail by Google

Attachment A

Mark Rees <mrees@fairhaven-ma.gov>

revised response to BU air paper

1 message

Lederer, Dave <Lederer.Dave@epa.gov> Wed, Dec 11, 2019 at 2:43 PM
To: "michele.paul.newbedford-ma.gov" <michele.paul@newbedford-ma.gov>, Mark Rees <mrees@fairhaven-ma.gov>

Below is the revised version of EPA "desk statement" regarding BU paper on potential risk.

In short: The most significant risk of PCB exposure at the NBH Superfund Site is from consumption seafood caught in vicinity of the source of contamination, the NBH cleanup is steadily lowering exposures to PCBs in air, and the potential risks identified due to air exposures in the paper are unlikely due to the conservatism of the approach.

Thanks. Let me know if you have any further questions

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Desk Statement:

EPA has been monitoring airborne PCBs in and around New Bedford Harbor since the late 1980s. The measured levels of airborne PCBs have never exceeded EPA's health-based criteria. EPA will continue to closely review our ongoing monthly air sampling results and will take action to protect public health if necessary.

When discussing the risks of exposure to PCBs related to New Bedford Harbor, by far the largest health risk posed is consumption of locally-caught seafood from the area closest to the sources of PCB contamination.

EPA concurs with one of the main conclusions of the paper regarding the harbor cleanup, which is that EPA's Superfund cleanup "over the last ten years has actually reduced emissions of airborne PCBs." The paper also states, "As the PCB cleanup continues and the major source of PCBs is removed, it is expected that exposures to PCBs in ambient air will decrease with time." Finally, the paper states that active dredging does not appear to affect airborne PCBs levels in New Bedford.

The paper derives Margin of Exposure values (similar to safety margins) ranging from 20 to 10,000, indicating that the air concentrations near the harbor were 20 to 10,000 times lower than the lowest potential effect concentration for the thyroid. In the evaluation of multiple sources of uncertainty, the paper concludes that "In all cases, the implication would be that risk is overestimated."

EPA remains firmly committed to our work in New Bedford. We are proud that our work to dredge contaminated sediment from New Bedford Harbor is nearly complete. PCB levels in the Upper Harbor are now 100-times lower than they were previously and have been reduced to well below the cleanup level of 10 parts per million. EPA's cleanup work has already significantly improved – and will continue to further improve – the environment around New Bedford Harbor.

David Lederer

Team Leader,

New Bedford Harbor Superfund Site

US Environmental Protection Agency, Region I

New Bedford Harbor Superfund Site

857-274-7256 (cell)

617-918-1325 (office)

Science of the Total Environment xxx (xxxx) xxx

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Human health risks due to airborne polychlorinated biphenyls are highest in New Bedford Harbor communities living closest to the harbor

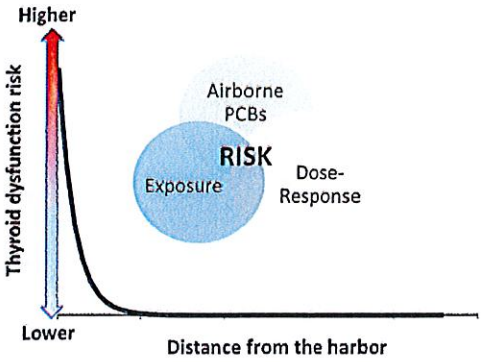
Wendy J. Heiger-Bernays [a,*], Kathryn Scott Tomsho [a], Komal Basra [a], Zoe E. Petropoulos [a], Kathryn Crawford [a], Andres Martinez [b], Keri C. Hornbuckle [b], Madeleine K. Scammell [a]

[a] Boston University School of Public Health, Department of Environmental Health, 715 Albany St., Talbot Building, Boston, MA 02118, United States
[b] Department of Civil & Environmental Engineering, IIHR-Hydroscience and Engineering, 4105 Seamans Center for the Engineering Arts and Sciences, The University of Iowa, Iowa City, IA 52242, United States

HIGHLIGHTS

- Community concerns focus on health risks associated with PCBs in the air.
- PCBs were highest in ambient air closest to the New Bedford Harbor Superfund Site.
- Toxicological data were used to derive a thyroid-based effect level for a Margin of Exposure (MOE) evaluation.
- Health risks are highest among people living adjacent to New Bedford Harbor.

GRAPHICAL ABSTRACT



ARTICLE INFO

Article history:
Received 18 June 2019
Received in revised form 12 November 2019
Accepted 15 November 2019
Available online xxxx

Editor: Adrian Covaci

Keywords:
Superfund
Polychlorinated biphenyls
Ambient air
Margin of Exposure
PCB congener
Thyroid

ABSTRACT

In response to concerns raised by communities surrounding the New Bedford Harbor Superfund site, we completed a field and modeling study that concluded the harbor is the primary source of polychlorinated biphenyls (PCBs) in air around the harbor. The follow-up question from residents was whether the PCBs measured in air pose a risk to their health. The US Environmental Protection Agency focuses their site-specific, risk-based decisions for site clean-up on cancers. We focused our assessment on the non-cancer effects on the thyroid based on the congener specific patterns and concentrations of PCBs measured in air near and distant to the harbor. Human and animal studies of PCB-induced effects on the thyroid provide evidence to support our analysis. Drawing from the published toxicological data, we used a Margin of Exposure (MOE) approach to derive a human-equivalent concentration in air associated with human health effects on the thyroid. Based on the MOEs calculated herein, evaluation of the MOE indicates that changes in thyroid hormone levels are possible among people living adjacent to the Harbor. Changes in thyroid hormone levels are more likely among people who live near the harbor compared to residents living in areas distant from the harbor. This risk assessment documents potential health risks associated with proximity to a marine Superfund Site using site-specific ambient air PCB congener data.

* Corresponding author.
E-mail address: whb@bu.edu (W.J. Heiger-Bernays).

https://doi.org/10.1016/j.scitotenv.2019.135576
0048-9697/

1. Introduction

New Bedford Harbor (NBH), located in southeastern Massachusetts, is surrounded by the towns of Acushnet, Dartmouth, Fairhaven, and the city of New Bedford, which have a combined population of approximately 150,000 (Census Bureau, 2015). In 1982, the US Environmental Protection Agency (EPA) placed the 18,000-acre harbor on the National Priorities List due to high concentrations of PCBs and metals measured in the water and sediments. The PCBs in New Bedford Harbor originated from two electronic capacitor manufacturers—Aerovox Corporation and Cornell Dublier. The companies discharged PCB-containing waste into the harbor and local sewer system beginning in the early 1940s (Pesch et al., 2001). Since becoming a Superfund site, the harbor has undergone multiple federal and state dredging operations, including "hot spot" clean-up for the most contaminated sediments in the 1990s and the current hydraulic dredging that began in 2004, with major removal operations in 2016–2018 (U.S. Environmental Protection Agency, 2014). These dredging activities involve moving contaminated sediment from the bottom of the harbor to disposal facilities, including the on-site confined aquatic disposal (CAD) cells located in the inner harbor. Dredging activities introduce the potential for increased release of airborne PCBs and subsequent exposure through inhalation of ambient PCBs (Vorhees et al., 1997; Wilson, 2015). For this reason, EPA has been monitoring air concentrations of total PCBs near the harbor since 2004 and comparing these levels to EPA-derived screening levels based on cancer risks. As of April 2018, the EPA reports that all of their monitored concentrations have been below the risk-based screening levels (U.S. EPA, 2017). However, the EPA screening levels are not based on the congeners measured in the air and the monitoring methods do not report congener-specific data. Rather, EPA reports data for groups of PCBs known as homologues following 24-hour air monitoring, reflecting the groups, but not individual congeners. Furthermore, until 2016, the EPA air monitors were located for convenience or where concentrations were anticipated to be highest. These locations were not necessarily reflective of where people are likely to spend time or be exposed. Beginning in late 2016 and extending through late 2017, EPA placed air monitors in locations closer to where people spend time.

People living and working near New Bedford Harbor are potentially exposed to PCBs through contact with water and sediments in the harbor, air around the harbor, indoor air and indoor dust, and consumption of seafood caught from the harbor. Cancer risks associated with seafood consumption are relatively well-characterized and have been the basis for closures of areas to fishing and fish consumption (Basra et al., 2018). We assessed inhalation of PCBs in ambient air by NBH-area residents, both adults and children. The primary exposure medium is the surface water of New Bedford Harbor, from which PCB congeners are volatilized (present in gas phase) or aerosolized (suspended in water droplets or with particulate matter) into the surrounding air. While there are other sources of ambient PCBs around New Bedford Harbor, our field and modeling study showed that the harbor is the primary source of PCBs in air around the harbor (Martinez et al., 2017).

We are now able to estimate residential exposures and health risk for residents of the towns and cities surrounding New Bedford Harbor: Acushnet, Dartmouth, Fairhaven and New Bedford. We initially sought to assess risk using the approach used by regulatory authorities for decision-making, which would allow for comparison of risk from consumption of PCB-contaminated fish with inhalation of PCBs in air.

While this is possible for assessment of cancer risk, it is not possible for non-cancer risk due to the lack of a reference concentration (RfC) (U.S. Environmental Protection Agency, 1994). EPA defines the RfC as the concentration of PCBs in air that sensitive populations can breathe all day of every day and not expect to experience ill health related to PCB exposure. The need for a PCB RfC has been documented, along with the acknowledgement of need for more research to form the scientific foundation of a RfC (Lehmann et al., 2015).

There is strong evidence supporting the thyroid as a target of PCB toxicity. We relied on the existing toxicological literature from which we derived a human equivalent concentration (HEC) of the airborne PCBs associated with adverse effects in rodent studies. We then compared the HEC with the ambient air concentrations of PCBs measured near homes around NBH to estimate a Margin of Exposure (MOE) for inhalation to PCBs at the Superfund Site. Rather than establishing a concentration deemed acceptable, such as a RfC, the MOE tells us about the likelihood of exposure to cause "unreasonable" risk (U.S. Environmental Protection Agency, 2012). It is a ratio of the toxicity effect level to the estimated exposure dose. The specific objectives of this analysis were as follows:

- Determine which PCB congeners are prevalent in ambient air measured around NBH and assess exposure to residents of surrounding communities (exposure assessment)
- Provide evidence for the thyroid as a target organ for PCB toxicity for the most prevalent congeners and two relevant Aroclors in the NBH region and/or lower-chlorinated, inhaled PCB congeners (hazard assessment)
- Calculate Margin of Exposure for residents (risk assessment).

2. Materials and methods

2.1. Exposure assessment

In response to requests from residents to monitor the air closer to where they work and live, the Boston University Superfund Research Program (BUSRP) and our Community Engagement Core partner Toxics Action Center worked with the Iowa Superfund Research Program (ISRP) and residents to identify locations and design a monitoring program to assess congener-specific ambient PCB concentrations in the air around New Bedford Harbor (Fig. S1). While EPA monitors airborne PCBs using high volume sampling for 24 hour periods, we used passive air samplers that integrate air concentrations over four to six weeks. We conducted four rounds of air monitoring in 2015 and 2016, one of which occurred during a period of hydraulic dredging and has not been previously reported. Details on sampling, analysis and community engagement methods are described elsewhere (BU Superfund Research Program, 2015; Martinez et al., 2017; Tomsho et al., 2018). Our measurements show that concentrations did not differ significantly across sampling events. Congener-specific concentrations can be found in the Supporting Information. The full PCB congener dataset with associated metadata is available in a freely accessible data repository at https://doi.pangaea.de/10.1594/PANGAEA.902925 (Martinez et al., 2019).

2.2. Hazard assessment

The Margin-of Exposure analysis is based on changes in thyroid hormone levels as a function of exposure to PCB congeners around NBH, therefore, in the hazard assessment we support the basis for selection of the thyroid as a target of PCB toxicity. We provide evidence from the epidemiology literature and reviewed studies of oral exposure to PCBs relevant to NBH to assess the overall weight of evidence supporting a connection between PCB exposure and the health effects. We then identified key studies that rely on inhalation exposures to PCBs, especially those congeners most abundant in the air around NBH. From this literature, we selected the key studies from which a Point of Departure (POD) air concentration could be taken.

2.3. Margin of Exposure determination

Using the published toxicological literature, we evaluated the dose-response relationship between PCB congeners and thyroid-related adverse health effects and identified a Point of Departure (POD) from which we derive a human-equivalent concentration (POD_{HEC}) in air.

Please cite this article as: W.J. Heiger-Bernays, K.S. Tomsho, K. Basra, et al., Human health risks due to airborne polychlorinated biphenyls are highest in New Bedford Harbor commu..., Science of the Total Environment, https://doi.org/10.1016/j.scitotenv.2019.135576

The POD is defined as the point on a dose-response curve established from toxicological or epidemiologic data that corresponds to an estimated low effect level or no effect level, marking the beginning of extrapolation to a relevant human dose or concentration. The principal studies used to develop the dose-response curve for POD determination were selected using an approach similar to that described in *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (Canter et al., 1998; U.S. Environmental Protection Agency, 1994). Using EPA default assumptions, we convert the rodent-based POD to the human equivalent concentration (POD_{HEC}). The Margin of Exposure (MOE) (U.S. EPA, 2012) is then calculated as the ratio of the POD_{HEC} to the geometric mean of the measured ambient airborne PCB concentrations surrounding NBH.

3. Results

3.1. Exposure assessment

PCB concentration measurements were averaged over the four rounds of sampling to determine an exposure metric for each sampling location. Over the study period at the 18 sampling locations, total PCB air concentrations ranged from 0.4 ng/m^3 to 38.6 ng/m^3 with an average across sampling locations of 7.0 ng/m^3 (standard deviation: 9.3 ng/m^3) and a geometric mean of 3.1 ng/m^3. Using non-parametric statistical tests, we concluded that the median concentration of total PCBs at each site and overall was not significantly different ($p < 0.05$) across the four rounds of testing. Therefore, the four rounds were combined for the risk estimate calculations.

As the distance from the harbor increases, total ambient airborne PCB concentrations decrease out to 625 m (Fig. 1). The pattern illustrates exponential decrease due to dispersion. The arithmetic and geometric mean of the 11 sampling locations within 625 m of the harbor are 10.9 ng/m^3 and 8.1 ng/m^3, respectively. We consider a distance further than 625 m to be background concentrations of total PCBs since concentrations remain relatively flat between 625 and 4000 m. The arithmetic and geometric means of the seven sampling locations further than 625 m from NBH are 0.7 ng/m^3. We observed a decrease of the 12 most present congeners in the air, from "near" to "distant" sites, whereas PCB11 increased in the air (Fig. 2).

Our previous analysis of data collected in the first three rounds of this study found that the PCB concentrations measured at all 18 locations had a very strong Aroclor 1242/1016 signal (Martinez et al., 2017). This is consistent with the historical use of these two Aroclor mixtures in the vicinity The analysis also found that the Aroclor 1242/1016 signal was the most pronounced at locations closest to the harbor across all sampling rounds. Although all congeners were measured, we identified twelve dominant PCB congeners or congener groupings in air surrounding NBH. PCBs 4, 6, 8, 17, 18 + 30, 20 + 28, 25, 26 + 29, 31, 44 + 47 + 65, 49 + 69 and 52 account for, on average, 63% of the total PCB ambient air exposure at NBH (54% minimum, 71% maximum). The relationship between dominant congeners and distance to the shoreline of NBH is shown in Fig. 3. Overall there does not appear to be a relationship between dominant congeners/congener groupings as percent of total PCBs, and distance to the shoreline of NBH. There is very little spatial variation in relative abundance of PCBs between sampling locations, suggesting that all twelve of these congeners/congener groupings are important contributors. It is noteworthy that the relative contribution of PCB11 to total PCBs *increases* with distance from NBH unlike the other congeners shown. Subsequent drops and spikes in the percent of PCB11 to total PCBs results in relative increases and decreases of the other twelve congeners/congener groupings. The presence of PCB11 at these distances may be due to its presence in, and volatilization from, modern paint pigment (Hu et al., 2008; Jahnke and Hornbuckle, 2019). NBH does not appear to be a source of PCB11. These findings provide evidence that these twelve congeners or congener groupings are important tracers of PCBs released from NBH, and may also be tracers of PCBs associated with production and use of electronic capacitors, the original source of PCBs in NBH.

Analysis of the air monitoring data suggest that PCBs are correlated with distance to the harbor and that concentrations significantly leveled off, reaching background levels, at approximately 625 m from the harbor. Therefore, we conducted the risk assessment with two separate exposure areas, dividing the sampling locations into those near to the harbor (<625 m from the harbor) and those distant to the harbor (>625 m from the harbor). Separate risk calculations were conducted for these two areas, based on the geometric means and are referred to as "near" and "distant".

3.2. Hazard assessment

To derive a relevant Point of Departure (POD) from which to estimate health risk, we identified evidence from the epidemiology and toxicological studies that support the thyroid as a target organ of PCB toxicity, with a focus on PCB congeners or mixtures relevant to those measured in the air at NBH. The availability of toxicological studies that are conducted with human-relevant mixtures of PCBs in air is limited, although there are multiple in vivo oral exposure and human epidemiological studies that are used to provide supporting evidence of an association between PCB exposure and adverse health effects.

Evidence is accumulating for a thyroid-dependent mechanism for effects of PCBs on the developing hypothalamic neuroendocrine systems (reviewed in Gauger et al., 2007 and Gore et al., 2019). Epidemiologic studies provide evidence of association between reduced thyroid hormone levels, or impaired thyroid hormone action, in infants and

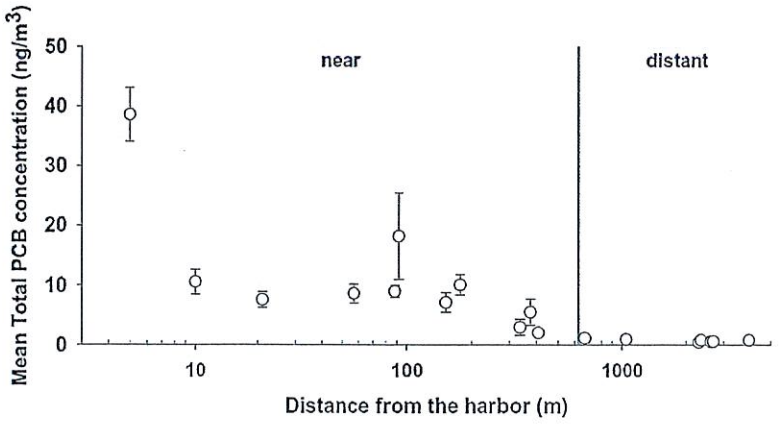


Fig. 1. Relationship between total PCB ambient air concentrations (ng/m^3) and distance (m) from New Bedford Harbor.

Please cite this article as: W.J. Heiger-Bernays, K.S. Tomsho, K. Basra, et al., Human health risks due to airborne polychlorinated biphenyls are highest in New Bedford Harbor commu..., Science of the Total Environment, https://doi.org/10.1016/j.scitotenv.2019.135576

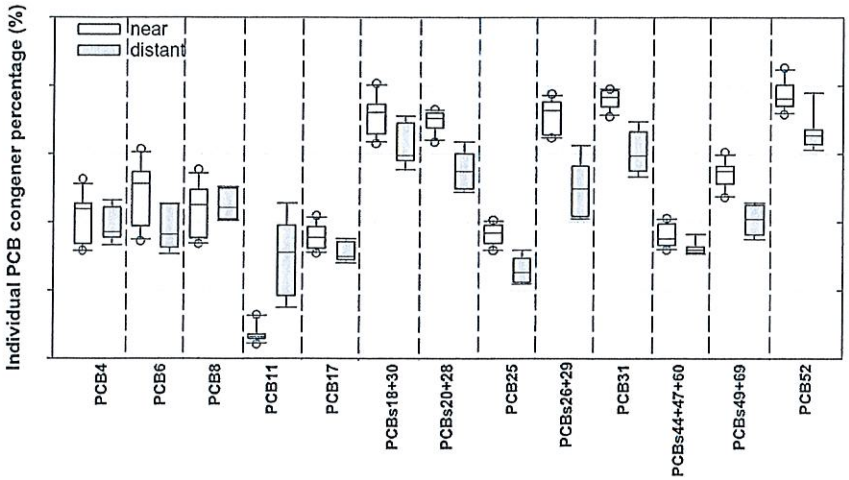


Fig. 2. Comparison of most dominant congeners/congener groupings as percentage of total PCBs at "near" and "distant" locations from New Bedford Harbor.

children as a result of prenatal or early life exposure (Curtis et al., 2019; Dallaire et al., 2008, 2009a, 2009b; Dirinck et al., 2016). As previously stated, EPA-derived screening levels used to assess PCBs near NBH are based on cancer risks. However, adverse health effects of PCB exposures, in addition to cancers have been extensively studied and evidence is available for outcomes that are influenced by thyroid hormone physiology, including impaired neurodevelopment (Forns et al., 2018; Grandjean et al., 2001; Sagiv et al., 2012; Verner et al., 2015), increase in metabolic diseases (Aminov et al., 2016b; Zani et al., 2019) and decreased birthweight (Govarts et al., 2012). Commonly observed health outcomes in epidemiologic studies, including those in which inhalation is a documented route of exposure to mixtures of PCBs, support endocrine-related outcomes including diabetes (Aminov et al., 2016a; Jorgensen et al., 2008; Lee et al., 2010; Philibert et al., 2009; Rylander et al., 2015; Wang et al., 2008), and altered thyroid volume and function (Alvarez-Pedrerol et al., 2008; Darnerud et al., 2010; Langer et al., 2007; Leijs et al., 2012; Sandau et al., 2002; Turyk et al., 2006). A number of these toxicants may also interfere with the hypothalamic–pituitary–thyroid regulatory axis and be associated with a reduced serum thyroxine (T4 or T3) concentration, but with a normal range of thyroid stimulating hormone (TSH). Animal and epidemiologic studies that examine brain development, indicate the potential for more subtle disruption of local thyroid hormone production or action that may be difficult to detect based on circulating thyroid hormone levels (Brent, 2010). The thyroid is a target organ of PCB toxicity, as documented by an expanding body of evidence that demonstrates that PCBs and other organochlorines influence thyroid function.

There is also a body of literature providing evidence that several environmental toxicants including PCBs can interfere with thyroid hormone, resulting in elevation in serum TSH or a reduction in serum T4 or T3 in animal models (Bansal and Zoeller, 2008; Lau et al., 2017; Martin and Klaassen, 2010). A number of these toxicants may also interfere with the hypothalamic–pituitary–thyroid regulatory axis and be associated with a reduced serum T3 or T4 concentration, but with a normal range of TSH.

Most of the animal studies of lower chlorinated congeners and thyroid effects have been conducted through oral administration of Aroclors. Of the twelve most dominant congeners in NBH air samples, only PCB congeners 28 and 52 have been studied in the context of thyroid effects. Most relevant oral exposure studies have been conducted using mixtures of lower chlorinated PCBs: Aroclor 1016, Aroclor 1221, and Aroclor 1242 and we present some of the evidence for PCBs effects on thyroid here. Of the papers that examined changes in thyroid hormone levels, only one examined thyroid stimulating hormone (TSH), a biologically relevant measure of thyroid function (Martin and Klaassen, 2010). Mayes et al. (1998) administered Aroclors 1016, 1242, 1254, and 1260 for 24 months to male and female Sprague-Dawley rats. Aroclors were suspended in hexane, combined with a small amount of diet (the "premix"). The hexane was evaporated, and the premix was blended with diet to achieve the desired final PCB

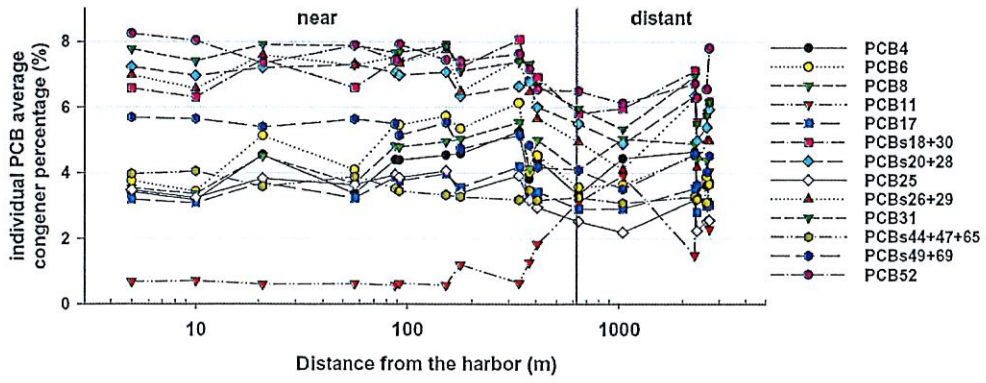


Fig. 3. Relationship between most dominant congeners/congener groupings as percent of total PCBs and distance from New Bedford Harbor.

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concentrations ranging from 25 to 200 ppm. The authors report that the thyroid glands of male animals receiving Aroclors 1242, 1254, and 1260 were enlarged in a non-dose-related manner. This study showed a dose-dependent incidence of thyroid gland follicular cell adenomas in males for Aroclors 1242, 1254, and 1260, with the incidence being uniform across dose groups and Aroclor mixtures. While the authors report that the neoplasms are likely due to hypersecretion of TSH, they do not discuss the possibility that changes in circulating levels of thyroid hormones may occur as a direct effect of the PCBs administered.

Crofton et al. (2005) dosed 23-day old female Long Evans rats by oral gavage for four consecutive days with 12 individual PCBs, including PCB-126, PCB-153, NBH-relevant PCB-28 and PCB-52, as well as mixtures of the 12. Serum total T4 was measured via radioimmunoassay in samples collected 24 h after the last dose. Doses of individual chemicals were associated with a 30% thyroid hormone decrease from control (ED30). In particular, both PCB-126 and PCB-153 demonstrated dose-dependent decreases in total T4, although PCB-126 was able to do this at a lower dose than PCB-153 and the mixture was shown to cause decreases in T_4 concentrations.

In order to determine the effects of individual PCB congeners on serum T3, T4 and TSH, Martin and Klaassen (2010) administered Aroclor 1254 or 1242, PCB-95, PCB-99, PCB-118 or PCB-126 in corn oil via oral gavage to male Sprague-Dawley rats for seven consecutive days. Rats were necropsied 24 h after the last dose. Serum total T4 and free T4 hormone levels were evaluated by radioimmunoassay and both were dramatically reduced in response to each of the seven treatments in a dose-dependent manner. Marked T4 reductions occurred in response to Aroclor 1254, PCB-99, and PCB-118. Serum TSH was not significantly affected by any of the compounds administered.

Martin et al. (2012) examined the effects of oral administration of 32 mg/kg-day of Aroclor 1242 to male Sprague-Dawley rats. Aroclor was administered via corn oil solution through oral gavage for seven consecutive days. Effects documented in the treated groups included decreases in serum thyroid hormones T4 and T3, although TSH was not measured.

Four in vivo rat inhalation studies evaluated Aroclor mixtures of direct relevance to NBH communities. One examined the uptake and elimination of Aroclor 1242 (Hu et al., 2010). Another (Casey et al., 1999), compared the adverse outcomes of inhalation versus ingestion of Aroclor 1242. The third, Hu et al. (2012), exposed female Sprague-Dawley rats via nose-only inhalation to a mixture of Aroclors 1242/1254 and PCB11 at an average air concentration of 520 ± 10 μg/m^3. The final inhalation-relevant study examined the difference in adverse outcomes of whole body exposure as compared to nose-only exposure to an Aroclor 1242/1254 + PCB11 mixture (Hu et al., 2015). Of the 12 most dominant congeners in NBH air samples, only PCB congeners 28 and 52 have associated toxicology studies.

Casey et al. (1999) compared whole body-inhalation of 0.9 μg/m^3 and oral administration of 0.436 ppm of Aroclor 1242 to male Sprague-Dawley rats over 30 days and examined the impacts on thyroid hormones and behavioral outcomes. Both exposure groups displayed decreases in rearing activity and ambulation and increases in T4 and T3 as compared with the control group, with the group exposed via inhalation showing larger increases as compared to the oral exposure group. Additionally, the group exposed via inhalation displayed an increase in intracellular vacuolization of follicular epithelial cells in the thyroid gland. TSH was not measured. Casey et al. (1999), reported elevated T3 and T4 accompanied by increased intracellular vacuolization of follicular epithelial cells of thyroid gland after inhalation exposure to 0.9 μg/m^3 volatilized Aroclor 1242. Casey et al. (1999) does not adequately describe exposure information including breathing zone concentrations and evidence supporting a uniform distribution of chemical within the exposure chamber. Furthermore, control animals were not exposed to air under the same conditions as animals exposed to PCBs by inhalation. Compared to animals in the control group, inhalation-exposed animals lived with reduced air flow (three changes of chamber air/h for exposed animals compared to fifteen for control animals) as well as increased humidity and temperature. These factors could all potentially contribute to differences in the baseline level of stress experienced by animals in these two groups, which could translate into differences in thyroid hormone levels and behavioral outcomes beyond those related to the PCB exposure. It is important to note that the animals may very well have been exposed to higher levels of PCBs than are reported by the study and may have been exposed not only by inhalation, but also by oral exposure as a result of grooming after PCB deposition on their fur.

In a short-term study intended to determine pharmacokinetic data for inhaled PCBs, Hu et al. (2010) administered Aroclor 1242 to male Sprague-Dawley rats in a nose-only inhalation system at 2.4 mg/m^3 for 2 h or at 8.2 mg/m^3 for 2 h each of 10 consecutive days. Pulmonary immune markers and PCB tissue levels were measured and demonstrated that the airborne PCB mixture contributed significantly to the body burden of lower-chlorinated congeners. Rats exposed in the multi-day study gained significantly less weight over the 10-day exposure period compared to sham-exposed animals. However, there were no significant changes observed in pulmonary immune markers. PCB levels were similar in lung, liver, and adipose tissue, lower in brain, and lowest in blood.

Hu et al. (2012) exposed female Sprague-Dawley rats via nose-only inhalation to a mixture of Aroclors 1242/1254 and PCB11 at an average air concentration of 520 ± 10 μg/m^3. The rats were exposed 2 h per day for the first week of exposure, and then 1.5 h per day for weeks 2–4. The dose was estimated to be 134 μg total PCBs per rat (reported as 446 μg/kg-day by Hu et al., 2015). The rats did not display any difference in their overall growth rate as compared to controls, nor an increase in enzymes that metabolize PCBs in the livers of the exposed animals, nor an increase in enzymes that metabolize PCBs in the livers of the exposed animals, nor a change in plasma thyroid hormone levels (as reported by Hu et al., 2015). In the 2012 study by Hu et al., female rats exposed to a dose of 446 μg/kg-b.w. were found to have no changes in weight gain or plasma thyroid hormone levels compared with control, although the authors. Reduced glutathione (GSH) is considered to be one of the most important scavengers of reactive oxygen species and its ratio with oxidized glutathione (GSSG) may be used as a marker of oxidative stress. The GSSG/GSH ratio was slightly elevated in the exposed group as compared to the controls, and the total hepatic glutathione was decreased (Hu et al., 2012).

Hu et al. (2015) exposed 11 week old female Sprague-Dawley rats through whole body and nose-only inhalation to a mixture of Aroclor 1242 and Aroclor 1254 supplemented with PCB 11 (64.0%:34.5%:1.5%, w/w). Nose-only exposed rats gained significantly less weight over the 28-day exposure period compared to controls. Plasma total thyroxine (T4) levels were significantly reduced in exposed animals compared to controls ($p = 0.0006$) and compared to both sham- and PCB-exposed female Sprague–Dawley rats. No change of free T4, total triiodothyronine (T3) or free T3 was observed. TSH levels were not measured. The nose-only group exposure was estimated at 1980 μg/kg-day and also displayed an increase in the formation of malondialdehyde (MDA) in the liver. The whole-body exposure was estimated at 1320 μg/kg-day and displayed paler cytoplasm in centrilobular hepatocytes as compared to the periportal hepatocytes compared with the nose-only exposures (Hu et al., 2015).

Most studies are limited in scope to toxicodynamics and few examine both toxicokinetics and toxicodynamics of PCB mixtures. This is an important distinction when attempting to understand the interaction of PCBs with the multiple components of the thyroid signaling pathways. Of the relevant toxicological literature, only four of the in vivo animal studies administered the PCBs via inhalation (Casey et al., 1999; Hu et al., 2010, 2012, 2015). Of the relevant congeners in NBH air, those most studied were PCB 28 and PCB 52, both of which are considered to be indicator PCBs because of their prevalence in both environmental samples and human biospecimens (Boalt et al., 2013). We conclude that changes in thyroid hormone levels in response to PCBs reported in the inhalation studies are supported by observations made in epidemiologic

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studies and in toxicological studies of oral exposure, which identify the thyroid gland as a target organ for PCB toxicity.

3.3. Margin of Exposure estimates

We used a Margin of Exposure approach to determine the health risk to humans exposed to airborne PCBs from NBH. This margin relates the range of concentrations shown to cause adverse effects in animal models with airborne PCB concentrations measured in the "near" and "distant" areas surrounding New Bedford Harbor. MOEs are calculated as the ratio of a POD (e.g., Lowest Observable Adverse Effect Level) to an exposure concentration (e.g., a representative concentration of airborne PCBs surrounding NBH). Large MOEs, those that are several orders of magnitude of difference between the POD and the exposure concentrations indicate less concern that current exposure levels may be causing adverse health effects in the exposed population. Smaller MOEs indicate a greater level of concern that adverse effects may occur.

Using an approach similar to that described in *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (Canter et al., 1998; U.S. Environmental Protection Agency, 1994), we identified the POD based on the principal studies relevant to airborne PCB exposure and adverse effects on the thyroid, which included Casey et al. (1999), Hu et al. (2012), and Hu et al. (2015). Effects on thyroid hormone levels were found to be the most consistent in inhalation studies and were well-supported by the results of oral exposure studies; thus, PODs based on changes in thyroid hormone levels were used to estimate the MOEs. While each of these studies has limitations (discussed above and in the Discussion section), nevertheless, all three studies identified changes to the thyroid, which supports the thyroid as sensitive to disruption by airborne PCBs.

These studies were selected because they are most relevant to community inhalation of PCBs in ambient air at NBH. The concentrations (in air) used in these studies range from [(0.9 μg/m^3) to (520 ± 10 μg/m^3–533 μg/m^3)] and reflect LOAELs as the PODs since effects on the thyroid in exposed versus unexposed animals were observed at all experimental airborne PCB concentrations (except in Hu et al., 2012). We derived a range of human equivalent PODs (POD_{HEC}) from the PODs identified in the animal studies, as shown in Table S2. These range from 0.9 μg/m^3 as the minimum POD_{HEC} ($POD_{HEC\ 1}$) to the maximum POD_{HEC} ($POD_{HEC\ 2}$) of 76 μg/m^3. Additionally, we chose not to rely on data from Hu et al. (2010) because this study used high doses of PCBs and very short exposure periods, neither of which are representative of airborne PCBs surrounding NBH.

The Margin of Exposure (MOE) is determined by dividing a Point of Departure (POD), derived from dose–response data by human exposure concentrations using the following equation:

$$MOE = PODHEC \div PCBNBH\ Ambient\ Air$$

As human exposure concentration declines, MOEs become larger, likewise, as human exposure concentration increases, MOEs become smaller. The "near" and "distant" designations are described previously and reflect the concentrations of total PCBs measured in air as a function of geographic distance from NBH. The MOEs generated for the lower and upper values from the ranges of POD_{HECs} and exposure concentrations for the "near" and "distant" areas at New Bedford Harbor are presented in Fig. 4 to provide a range of risks posed to humans exposed to airborne PCBs from NBH. As the POD_{HEC} increases, the MOE increases yet as the exposure concentration increases, the MOE decreases. The minimum POD_{HEC} ($POD_{HEC\ 1}$) with the maximum PCB concentration measured shows an MOE of 20, just over one order of magnitude higher relative to "1". Additionally, the maximum POD_{HEC} ($POD_{HEC\ 2}$) compared with the minimum PCB concentration measured shows an MOE of 10,000.

4. Discussion

Based on the concentrations of airborne PCBs measured around NBH, decreases in thyroid hormone levels are possible in people living adjacent to the harbor, particularly in "near" harbor locations. Thyroid hormones are essential for normal behavioral, intellectual, and neurologic development and inadequate levels of these hormones has a negative effect on brain development. In a cohort of children born to mothers residing adjacent to NBH, moderate associations between PCB and *p,p'*-DDE levels and Activity Deficit Hypertension Disorder (ADHD)-like behaviors have been reported (Sagiv et al., 2010). Our findings are based on: (1) robust, empirically-measured exposure concentrations and (2) less certain toxicological evidence from published studies. We compared measured concentrations of PCBs in air around NBH with estimated concentrations of PCBs that are associated with adverse outcomes in previously published literature. The scientific evidence to date strongly supports changes to thyroid hormone levels as an important mode of action by which these (and the more heavily chlorinated) PCBs act on mammalian systems (Miller et al., 2009). While a more detailed evaluation of the adverse outcome pathway (s) for inhaled PCBs is warranted, it is beyond the scope of this paper.

The dose-response calculations were subject to key limitations that contribute uncertainty to the MOEs. The most important limitation is the selected toxicological studies. While the study by Hu et al. (2012) is methodologically stronger than the study by Casey et al. (1999) it

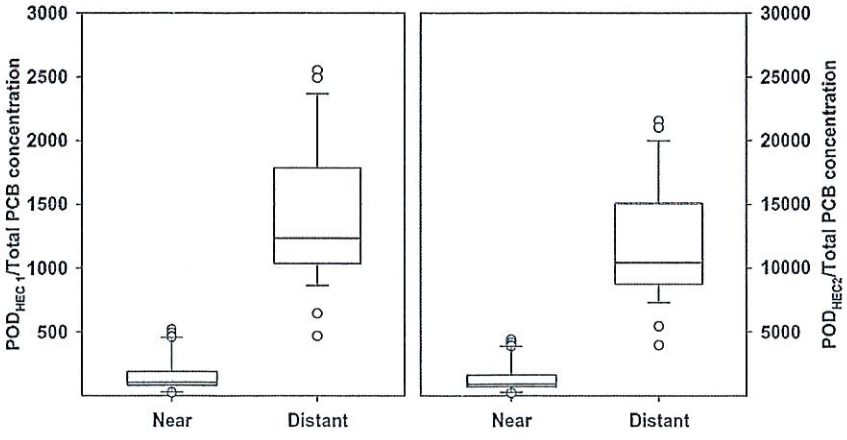

Fig. 4. Margin of Exposure (MOE) at "near" and "distant" locations. Left panel: $POD_{HEC\ 1}$ and right panel: $POD_{HEC\ 2}$.

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did not identify changes in thyroid hormones levels. Casey et al. (1999) demonstrate an increase in T3 and T4 levels in adolescent rats, rather than a decrease as has been reported in the other studies. Elevation in these hormone levels may be transient, as suggested by the authors. Had TSH been measured, more information about the thyroid hormone regulation could have been determined. The implications of elevated thyroid hormones would be borne out in observed rates of hyperthyroidism, or transient increases in T3/T4 and may not be as severe in the developing fetus or child. In contrast, Hu et al. (2015) does identify decreases in T4 levels compared with controls, suggesting that the reduction in T4 level may be associated with a PCB dose level between the doses of 446 and 1320 μg/kg body weight. All of the studies rely on limited dose ranges and durations that may be less accurate than reported. We used a default dosimetric adjustment factor for respiratory tract region for a complex mixture that may be an aerosol, rather than a gas. By using the default adjustment factor, we may have overestimated the POD_{HEC}, resulting in an overestimation of the MOE. In addition, the PCB concentration reported by Hu et al. (2012) may have been underestimated, in which case the resultant POD_{HEC} would be lower, resulting in a lower MOE. The lowest POD identified may in fact not be a LOAEL, but a NOAEL, because the dose at which this occurs did not result in a change in plasma thyroid hormone levels and the POD from Hu et al. (2015) has less uncertainty associated with it. In all cases, the implication would be that risk is over-estimated. Our straight-forward approach to derivation provides a starting point for a more data-driven POD that could be based on a Benchmark Dose or one that uses pharmacokinetic data to better estimate a POD. In addition, existing body burdens of PCBs should also be considered as an on-going source of internal exposure, as should consumption of foods contaminated with PCBs. Human exposures to PCBs in air occur through inhalation and dermal contact (Weschler and Nazaroff, 2012) and PCBs are absorbed through the skin (Garner and Matthews, 1998). It is beyond the scope of this analysis to evaluate the predicted uptake through skin based on simple pharmacokinetic models, but the total uptake of PCBs from the ambient air may be under-estimated for people living adjacent to NBH for decades.

We have shown the relevance of non-cancer outcomes for PCBs, and yet we are unable to assess risk using EPA methodologies because there is no non-cancer toxicity value (RfC) published by the US EPA for inhalation of PCBs. The exposure calculations in this risk assessment were subject to key limitations that contribute uncertainty to the estimates. First, the exposure calculations are based on the assumption that concentrations of ambient PCBs around New Bedford Harbor are constant over time. However, if concentrations decrease over time as a result of clean-up of the harbor, the risk estimates in this assessment will be overestimates. Conversely, if remediation activities create additional exposures, these will be underestimates. There is evidence of the former, but not the latter. Active dredging does not appear to affect airborne PCBs emitted from NBH. We measured airborne PCBs were measured during active dredging during the fourth four-week sampling period (July–August 2016) and they were not statistically different from those measured in air samples collected during the previous three sampling periods (July–November 2015). This finding begins to address community concerns about air emissions during this period of dredging (Tomsho et al., 2018). Our modeling studies indicate that removal of contaminated sediment over the last ten years has actually reduced emissions of airborne PCBs (Martinez et al., 2017).

Exposure estimates for individuals living farther than 2700 m from the harbor are not assessed in this risk assessment. However, the risk for these individuals (based on exposure to PCBs from the harbor alone) is unlikely to be greater than the estimates described for people living near the harbor given the concentrations of PCBs reported previously (Martinez et al., 2017). While most individuals spend approximately 80–90% of their day indoors (U.S. EPA, 2011), we assume that people were exposed to outdoor air concentrations every day for 24 h each day. However, this assumption is not necessarily an overestimate of exposure, since indoor and ambient air exchange is highly variable and difficult to predict (Weschler and Nazaroff, 2008; Yamamoto et al., 2010). We recognize that this assessment is not a cumulative risk assessment as described by Payne-Sturges et al. (2015) and therefore does not evaluate the full suite of stressors impacting the residents living and working in this Environmental Justice community adjacent to the harbor.

A major gap in the assessment of risk posed by PCBs is the lack of a RfC used for evaluation of non-cancer outcomes from inhalation of PCBs. In lieu of the RfC, we calculated the MOE for people living near NBH. While this risk assessment is limited in scope, we have attempted to use a site-specific, data-driven approach to calculating our MOE. The strengths of the calculated MOEs includes the relevance and study design of the critical studies on which the POD_{HECs} are based and the data-driven exposure assessment. Notably, we incorporated previously unavailable ambient air concentrations of PCB congeners and used longer duration of residence reflective of many local residents. Our analysis focused on the subset of PCB congeners most prevalent in air samples collected from around NBH. However, prevalence by weight does not necessarily mean that potential health impacts are limited to these congeners. This is especially relevant since existing body burdens of bioaccumulated PCBs and ongoing exposures through diet may play an integral role in the physiological response to additional inhalation exposure to PCBs. Despite these limitations, we show that non-cancer outcomes, specifically changes in thyroid hormone levels are possible in people who reside closest to the harbor. As NBH clean-up continues and the major source of PCBs is removed, it is expected that exposures to PCBs in ambient air will decrease with time.

5. Conclusion

Residents living near New Bedford Harbor are exposed to PCBs through multiple pathways, including consumption of fish and shellfish caught from the harbor, inhalation of volatile PCB congeners in ambient (outdoor) air, and possibly via indoor air. This risk assessment estimates the health risk to people living around New Bedford Harbor with a focus on ambient air exposures (Martinez et al., 2017). Specifically, PCB congeners that are present in ambient air in NBH communities may result in thyroid hormone level changes in response to inhaled PCBs. The estimated MOEs closest to NBH indicate the potential for a community at risk of thyroid mediated outcomes. This finding is consistent with observations made in previously published epidemiologic studies, which identify the thyroid as a target organ for PCB toxicity.

Declaration of competing interest

All of the authors declare that they do not have any actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations within three years of beginning the submitted work that could inappropriately influence, or be perceived to influence this work.

Acknowledgements

The authors thank Karen Vilandry of Hands Across the River Coalition (HARC) and the residents of New Bedford, Fairhaven, Dartmouth, and Acushnet, MA, who hosted the air samplers on their properties and who actively participated in this work. We thank Sylvia Broude and Claire Miller with Toxics Action Center who helped recruit and communicate with residents throughout the project, and attorneys Staci Rubin and Richard Juang, formerly/representing Alternatives for Community and Environment. We thank D. Lederer at US EPA Region 1 and P. Craffey at MassDEP for sharing information about the dredging processes and data related to the NBH Superfund Site, and the following reviewers of our data and analyses, listed in alphabetical order of last name: David Carpenter, PhD, Stephen Lester, MSc, Harlee Strauss, PhD.

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Funding

This study was supported by grants from the National Institute of Environmental Health Sciences to the Boston University Superfund Research Program NIEHS/NIH P42 ES007381-19S1 and the University of Iowa Superfund Research Program NIEHS/NIH P42 ES013661. ZEP and KB are supported by a grant from the National Institute of Environmental Health Sciences (T32 ES014562).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2019.135576.

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Please cite this article as: W.J. Heiger-Bernays, K.S. Tomsho, K. Basra, et al., Human health risks due to airborne polychlorinated biphenyls are highest in New Bedford Harbor commu..., Science of the Total Environment, https://doi.org/10.1016/j.scitotenv.2019.135576

Gmail by Google

Mark Rees <mrees@fairhaven-ma.gov>

BU meeting reminder

1 message

Hands Across the River Coalition, Inc. <harcgnb@gmail.com> Mon, Dec 16, 2019 at 1:23 PM
Bcc: mrees@fairhaven-ma.gov

Good afternoon,

Just a reminder that the Boston University, BU, leading scientists and author of the recent report on health risks from airborne PCBs from the New Bedford Harbor Superfund site, will be at our HARC meeting on Thursday, this week, December 19, 2019.

This is an informal meeting for local residents in New Bedford, Acushnet, Fairhaven, and Dartmouth who may be affected by airborne PCBs from the harbor.

The meeting will be held at the New Bedford Main Library, 613 Pleasant Street, New Bedford, corner of William Street, at 6:00 PM - 8:30 PM, in the 3rd floor auditorium.

This is your opportunity to ask them more about the health risks from breathing in this PCB contaminated air.

Please attend.

Regards,
Karen Vilandry
President
Hands Across the River Coalition Inc

508.951.1184

Attachment B

Date: December 16, 2019

To: Public officials in Fairhaven, Mattapoisett, Marion, Wareham
From: Mattapoisett Resident Bonne DeSousa

Re: SRPEDD strategy for Route 6

Dear public servants and committee members,

In summer 2019 our regional planning agency, SRPEDD, conducted public hearings about Route 6 between Fairhaven and Wareham. Residents and officials told planners about their experience and concerns. Among the concerns for this important, historic and scenic regional connector:

- Traffic is too fast.
- Walking on the sidewalk is "harrowing" and unpleasant.
- There are too few safe pedestrian crossings.
- Traffic speeds and patterns in established business districts should change to promote a thriving business climate.
- The narrow shoulders with hard cub are dangerous.
- There are several problem areas of poor drainage.
- Sidewalks are filled with obstructions.

On Dec 11, 2019 in the first of two public hearings whose stated purpose is to arrive at consensus, SRPEDD planners presented four one-size-fits-all alternatives that rated vehicle waiting times at specific signalized intersections. Those in attendance learned that for any scenario, only a few intersections are likely to receive a failing score 20 years and 40 years into the future. The presentation also recommended geometry changes to several un-signalized intersections. As to resident concerns, the assembled audience of residents, road users and public servants were told that the stated concerns can't be satisfied with current design guidelines given the layout width and the 85% rule for setting speeds, so they weren't addressed. All four strategies SRPEDD presented are inadequate because they rely ONLY on design solutions that exist in the DOT regulatory manuals.

I hope that public officials will ask for a fifth consensus strategy that addresses the resident concerns. Mr. Cornock said this would mean a DOT design exception process must be engaged. So be it. Towns should ask that another public hearing occur when our seasonal residents return to review the fifth consensus strategy.

There are at least 10 segments of Route 6 with specific character: Business districts, school zones, dense residential neighborhoods, high traffic intersections, scenic views and protected lands, frequent curb cuts for single family homes. Each of these is unique. Because future development is not likely to move traffic flow over capacity, then the biggest effort for planning should focus on user concerns according to the specific character of land use along the corridor. Please ask SRPEDD for a fifth strategy with variable lane configurations and descriptive design

goals for segments to be studied for traffic calming, drainage, wider shoulders, pedestrian crossings, center turning lanes.

Town boards, committees and planners need to make their preference known and can do so by going to the next public hearing January 6, 2020 at Sippican School in Marion 6-8PM, or by filling in comment cards, or emailing SRPEDD and saying we want a 5th alternative that addresses user needs in context-specific ways. Web based Comment cards are available here: http://www.srpedd.org/Route-6-Corridor-Study#HowtoParticipate

Emails can be addressed to
jcornock@srpedd.org

The address for all hard copy letters is
c/o Jed Cornock
Project Manager SRPEDD
88 Broadway Taunton, MA
02780

Phone number for SRPEDD is 508 824

Sincerely,
Bonne DeSousa,

12 Marion Road, Mattapoisett 02739
508 951 2406

Attachment C

2020 RENEWALS
FOR APPROVAL BY THE BOARD OF SELECTMEN
December 16, 2019

***Pending Bldg.**

LIQUOR LICENSES, FAIRHAVEN, MA 02719

1. *Gene's Famous Seafood, 146 Huttleston Avenue, Fairhaven, MA
2. The Bitter End Lounge, 407-409 Huttleston Avenue, Fairhaven, MA
3. Frontera Grill, 214 Huttleston Avenue, Fairhaven, MA
4. Sweet Ginger Asian Cuisine & Bar, 179-181 Huttleston Avenue, Fairhaven, MA
5. Mike's Restaurant, 390 Huttleston Avenue, Fairhaven, MA
6. Dorothy Cox's Candies, 21 Berdon Way, Fairhaven, MA
7. *Wah May Restaurant, 51 Main Street, Fairhaven, MA
8. *Elisabeth's Restaurant, 1 Middle Street, Fairhaven, MA
9. 99 Restaurant & Pub, 32 Sconticut Neck Road, Fairhaven, MA
10. Southcoast Wine & Spirits, 355 Huttleston Avenue, Fairhaven, MA
11. Brick Pizzeria Napoletana, 213 Huttleston Avenue, Fairhaven, MA
12. *Minerva Pizza House, 75 Main Street, Fairhaven, MA
13. Paul's Sports Corner, 19 Howland Road, Fairhaven, MA
14. Connolly's Liquor Mart, 36 Howland Road, Fairhaven, MA
15. Old Oxford Pub, 346 Main Street, Fairhaven, MA
16. Fairhaven Wine & Spirits, 105 Sconticut Neck Road, Fairhaven, MA
17. Sivalai Thai Cuisine, 130 Sconticut Neck Road, Fairhaven, MA
18. M & J Fairhaven, Inc.-Riccardi's Restaurant, 1 David Drown Blvd., Fairhaven, MA
19. Bayside Lounge, 125 Sconticut Neck Road, Fairhaven, MA
20. Friendly Farm Convenience, 121 Sconticut Neck Road, Fairhaven, MA
21. Cardoza's Wine & Spirits, 6 Sconticut Neck Road, Fairhaven, MA
22. *Mackatan General Store, 39 Causeway Road, Fairhaven, MA
23. *Douglas Wine & Spirits, 1 Peoples Way, Fairhaven, MA
24. The Pasta House Restaurant, 100 Alden Road, Fairhaven, MA
25. *Fort Phoenix Post 2892, Veterans of Foreign Wars of USA, 109 Middle Street, Fairhaven, MA
26. *Acushnet River Safe Boating Club, 80 Middle Street, Fairhaven, MA
27. *Off The Hook, 56 Goulart Memorial Drive, Fairhaven, MA
28. Ice House, LLC, 136 Huttleston Avenue, Fairhaven, MA
29. *Seaport Inn, 110 Middle Street, Fairhaven, MA
30. *Vila Verde Restaurant, 362-364 Main Street, Fairhaven, MA
31. *Rasputin's Tavern, 122 Main Street, Fairhaven, MA
32. *Ocean State Job Lot, 11 Berdon Way, Fairhaven, MA
33. *Moriarty Liquors, 101 Middle Street, Fairhaven, MA

34. Town Crier, 5 Maitland Street, Fairhaven, MA
35. Courtyard Restaurant, 270 Huttleston Avenue, Fairhaven, MA
36. *The Ebb Tide, 47 Middle Street, Fairhaven, MA
37. Scuttlebutts Liquors, 407-409 Main Street, Fairhaven, MA
38. *Huttleston License, LLC-Stevie's-A Package Store, 115 Huttleston Avenue, Fairhaven, MA
39. *Cleary's Pub, 111 Huttleston Avenue, Fairhaven, MA
40. *Fairhaven Post No. 166, American Legion, 54 Main Street, Fairhaven, MA

COMMON VICTAULER LICENSES-FAIRHAVEN, MA 02719

1. Papa Gino Pizza, 171 Huttleston Avenue, Fairhaven, MA
2. Subway, 42 Fairhaven Commons Way, Fairhaven, MA
3. Taco Bell, 33 Alden Road, Fairhaven, MA
4. Subway, 20 Sconticut Neck Road, Fairhaven, MA
5. Burger King, 180 Huttleston Avenue, Fairhaven, MA
6. Tropical Smoothies Café, 29 Alden Road, Fairhaven, MA
7. McDonald's Restaurant, 14 Plaza Way, Fairhaven, MA
8. Wendy's Restaurant, 7 Fairhaven Commons Way, Fairhaven, MA
9. Mac's Soda Bar, 116 Sconticut Neck Road, Fairhaven, MA
10. Dunkin Donuts, 18 Plaza Way, Fairhaven, MA
11. Dunkin Donuts, 32 Howland Road, Fairhaven, MA
12. Little Village Café, 23 Center Street, Fairhaven, MA
13. Palace Pizza & More, 69 Huttleston Avenue, Fairhaven, MA
14. Galaxy Pizza, 342 Main Street, Fairhaven, MA
15. Scramblers Breakfast & Bagel, 2 Sconticut Neck Road, Fairhaven, MA
16. 7-Eleven, 188 Huttleston Avenue, Fairhaven, MA
17. Brady's Ice Box, 12 Ferry Street, Fairhaven, MA
18. Margaret's Restaurant, 16 Main Street, Fairhaven, MA
19. Jake's Diner, 104 Alden Road, Fairhaven, MA
20. Mystic Café, 398 Main Street, Fairhaven, MA
21. Flour Girls Baking, 230 Huttleston Avenue, Fairhaven, MA
22. The Nook Café, 58 Washington Street, Fairhaven, MA
23. Festira Buffet, 31 Berdon Way, Fairhaven, MA
24. Honey Dew Donuts, 87 Huttleston Avenue, Fairhaven, MA
25. Phoenix Restaurant, 140 Huttleston Avenue, Fairhaven, MA
26. Yia Yia's Pizza Café, 381 Sconticut Neck Road, Fairhaven, MA

CAR DEALER LICENSES, FAIRHAVEN, MA 02719

1. Fairhaven Gas, Inc., 134 Huttleston Avenue, Fairhaven, MA
2. Fairhaven Gas, Inc.-Valero's, 130 Huttleston Avenue, Fairhaven, MA
3. Howard's Auto Sales, 10 Arsene Way, Fairhaven, MA
4. Guard Enterprises, 110 Alden Road, Fairhaven, MA
5. Alden Buick GMC, 6 Whalers Way, Fairhaven, MA
6. Alden Mazda, 37 Alden Road, Fairhaven, MA
7. Artistic Auto Body & Auto Sales, 98 Middle Street, Fairhaven, MA
8. Sarkis Enterprises, Inc. (A & A Auto), 196 Huttleston Avenue, Fairhaven, MA
9. RRR Auto Sales, 372 Huttleston Avenue, Fairhaven, MA
10. First Hot Line Auto Sales, Inc.-Fairhaven Getty Auto Sales, 371 Huttleston Avenue, Fairhaven, MA

REPAIR LICENSES, FAIRHAVEN, MA 02719

1. Fairhaven Gas, Inc., 134 Huttleston Avenue, Fairhaven, MA
2. Guard Enterprises, 110 Alden Road, Fairhaven, MA
3. Alden Buick GMC, 6 Whalers Way, Fairhaven, MA
4. Alden Mazda, 37 Alden Road, Fairhaven, MA
5. Artistic Auto Body & Auto Sales, 98 Middle Street, Fairhaven, MA
6. Sarkis Enterprises, Inc.(A & A Auto), 196 Huttleston Avenue, Fairhaven, Mae
7. A-1 Crane Company, 86-88 Middle Street, Fairhaven, MA
8. *Aaron's Auto Glass, 232 Huttleston Avenue, Fairhaven, MA
9. Jiffy Lube #1229, 31 Alden Road, Fairhaven, MA
10. Automotive Diagnostic Service, 162 Sconticut Neck Rd., Fairhaven, MA
11. Dattco Sales & Services, 72 Sycamore Street, Fairhaven, MA
12. Nice N' Clean Car Wash, 320 Huttleston Avenue, Fairhaven, MA
13. Rick's Services, 241 R. Huttleston Avenue, Fairhaven, MA
14. Manny's Service Station, 82 Bridge Street, Fairhaven, MA
15. Sullivan Tire Company, 9 Plaza Way, Fairhaven, MA
16. JR's Auto Shop, 276 Huttleston Avenue, Fairhaven, MA
17. Roland's Tire Service, 11 Howland Road, Fairhaven, MA
18. Jet Wash Car Was, 21 People's Way, Fairhaven, MA
19. RRR Auto Sales, 372 Huttleston Avenue, Fairhaven, MA
20. Leban Fuel, Inc., d/b/a Fairhaven Getty, 371 Huttleston Avenue, Fairhaven, MA

LODGING HOUSE LICENSE, FAIRHAVEN, MA 02719

1. Kopper Kettle Guest House, 41 Huttleston Avenue, Fairhaven, MA
2. Delano Homestead Bed & Breakfast, 39 Walnut Street, Fairhaven, MA

PRIVATE LIVERY LICENSE, FAIRHAVEN, MA 02719

1. Elite Transportation, 1 Deerfield Lane, Fairhaven, MA

Gmail by Google

Attachment D

Mark Rees <mrees@fairhaven-ma.gov>

Fwd: West Island Causeway Beach

5 messages

Charles Murphy <cmurphy@fairhaven-ma.gov> Thu, Dec 5, 2019 at 1:04 AM
To: Mark Rees <mrees@fairhaven-ma.gov>

Sent from my iPhone

Begin forwarded message:

From: Lisa Esten <westisland2019@gmail.com>
Date: December 4, 2019 at 4:50:02 PM EST
To: cmurphy@fairhaven-ma.gov
Subject: West Island Causeway Beach

Fairhaven Board of Selectmen
40 Center Street
Fairhaven, MA 02719

RE: Ristuccia - West Island Causeway Beach

Dear Board of Selectmen Charlie Murphy,

On behalf of the West Island Improvement Association (WIIA), we are writing to express our disappointment over the recent position the board has taken relative to the Ristuccia beach located on the north side of Causeway Road.

As you may know, Mr. Ristuccia has offered to donate the north side beach to the WIIA once the title issue is resolved. In exchange, the WIIA will reimburse Mr. Ristuccia for his costs associated with clearing the title. It is our understanding that the Town and Mr. Ristuccia, through counsel, had earlier drafted an agreement that would avoid a lengthy and costly resolution through the Land Court. We are now told that the Town will not assist in resolving the title issue and as a result legal fees and associated costs to be paid by the WIIA will increase significantly.

We would ask that you reconsider your position in order to avoid costly legal fees for both parties through a Land Court trial. As the record reflects, the Town did not intend to take the north side beach some 15 years ago (see attached). Case law clearly supports that Mr. Ristuccia still owns the north side beach.

The WIIA is a nonprofit that promotes the wellbeing of the Island, supports and donates to other organizations and groups in Town, and annually awards scholarships to graduating high school seniors attending college. The potential for high legal expenses associated with clearing the title will impact the WIIA's ability to support established programs for the community.

Thank you for your consideration regarding this matter.

For the West Island Improvement Association Board of Directors,

Lisa Esten

President WIIA

2 attachments

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Attachment E

Part I ADMINISTRATION OF THE GOVERNMENT

Title XX PUBLIC SAFETY AND GOOD ORDER

Chapter 140 LICENSES

Section 157 NUISANCE OR DANGEROUS DOGS; ORDERS FOR REMEDIAL ACTION; APPEAL; VIOLATION OF ORDER

Section 157. (a) Any person may file a complaint in writing to the hearing authority that a dog owned or kept in the city or town is a nuisance dog or a dangerous dog; provided, however, that no dog shall be deemed dangerous: (i) solely based upon growling or barking or solely growling and barking; (ii) based upon the breed of the dog; or (iii) if the dog was reacting to another animal or to a person and the dog's reaction was not grossly disproportionate to any of the following circumstances:

(1) the dog was protecting or defending itself, its offspring, another domestic animal or a person from attack or assault;

(2) the person who was attacked or threatened by the dog was committing a crime upon the person or property of the owner or keeper of the dog;

(3) the person attacked or threatened by the dog was engaged in teasing, tormenting, battering, assaulting, injuring or otherwise provoking the dog; or

(4) at the time of the attack or threat, the person or animal that was attacked or threatened by the dog had breached an enclosure or structure in which the dog was kept apart from the public and such person or animal was not authorized by the owner of the premises to be within such enclosure including, but not limited to, a gated, fenced-in area if the gate was closed, whether locked or

unlocked; provided, however, that if a person is under the age of 7, it shall be a rebuttable presumption that such person was not committing a crime, provoking the dog or trespassing.

The hearing authority shall investigate or cause the investigation of the complaint, including an examination under oath of the complainant at a public hearing in the municipality to determine whether the dog is a nuisance dog or a dangerous dog. Based on credible evidence and testimony presented at the public hearing, the hearing authority shall: (i) if the dog is complained of as a nuisance dog, either dismiss the complaint or deem the dog a nuisance dog; or (ii) if the dog is complained of as a dangerous dog: (A) dismiss the complaint; (B) deem the dog a nuisance dog; or (C) deem the dog a dangerous dog.

(b) If the hearing authority deems a dog a nuisance dog, the hearing authority may further order that the owner or keeper of the dog take remedial action to ameliorate the cause of the nuisance behavior.

(c) If the hearing authority deems a dog a dangerous dog, the hearing authority shall order 1 or more of the following:

(i) that the dog be humanely restrained; provided, however, that no order shall provide that a dog deemed dangerous be chained, tethered or otherwise tied to an inanimate object including, but not limited to, a tree, post or building;

(ii) that the dog be confined to the premises of the keeper of the dog; provided, however, that "confined" shall mean securely confined indoors or confined outdoors in a securely enclosed and locked pen or dog run area upon the premises of the owner or keeper; provided further, that such pen or dog run shall have a secure roof and, if such enclosure has no floor secured to the sides thereof, the sides shall be embedded into the ground for not less than 2 feet; and provided further, that within the confines of such pen or dog run, a dog house or proper shelter from the elements shall be provided to protect the dog;

(iii) that when removed from the premises of the owner or the premises of the person keeping the dog, the dog shall be securely and humanely muzzled and restrained with a chain or other tethering device having a minimum tensile strength of 300 pounds and not exceeding 3 feet in length;

(iv) that the owner or keeper of the dog provide proof of insurance in an amount not less than $100,000 insuring the owner or keeper against any claim, loss, damage or injury to persons, domestic animals or property resulting from the acts, whether intentional or unintentional, of the dog or proof that reasonable efforts were made to obtain such insurance if a policy has not been issued; provided, however, that if a policy of insurance has been issued, the owner or keeper shall produce such policy upon request of the hearing authority or a justice of the district court; and provided further, that if a policy has not been issued the owner or keeper shall produce proof of efforts to obtain such insurance;

(v) that the owner or keeper of the dog provide to the licensing authority or animal control officer or other entity identified in the order, information by which a dog may be identified, throughout its lifetime including, but not limited to, photographs, videos, veterinary examination, tattooing or microchip implantations or a combination of any such methods of identification;

(vi) that unless an owner or keeper of the dog provides evidence that a veterinarian is of the opinion the dog is unfit for alterations because of a medical condition, the owner or keeper of the dog shall cause the dog to be altered so that the dog shall not be reproductively intact; or

(vii) that the dog be humanely euthanized.

No order shall be issued directing that a dog deemed dangerous shall be removed from the town or city in which the owner of the dog resides. No city or town shall regulate dogs in a manner that is specific to breed.

(d) Within 10 days after an order issued under subsections (a) to (c), inclusive, the owner or keeper of a dog may bring a petition in the district court within the judicial district in which the order relative to the dog was issued or where the dog is owned or kept, addressed to the justice of the court, praying that the order be reviewed by the court or a magistrate of the court. After notice to all parties, the magistrate shall, under section 62C of chapter 221, review the order of the hearing authority, hear the witnesses and affirm the order unless it shall appear that it was made without proper cause or in bad faith, in which case the order shall be reversed. A party shall have the right to request a de novo hearing on the complaint before a justice of the court.

(e)(1) Pending an appeal by an owner or keeper under subsection (d), a hearing authority may file a petition in the district court to request an order of impoundment at a facility the municipality uses to shelter animals for a dog complained of as being a dangerous dog. A municipality shall not incur liability for failure to request impoundment of a dog under this subsection.

(2) A justice of a district court, upon probable cause to believe that a dog is a dangerous dog or that a dog is being kept in violation of this section or in violation of an order issued under this section by a hearing authority or a court, may issue an order: (i) of restraint; (ii) of confinement of the dog as considered necessary for the safety of other animals and the public; provided, however, that if an order of confinement is issued, the person to whom the order is issued shall confine the dog in accordance with clause (ii) of subsection (c); or (iii) of impoundment in a humane place of detention that the municipality uses to shelter animals; or (iv) any other action as the court deems necessary to protect other animals and the public from the dog.

(f) A justice of the district court shall hear, de novo, an appeal filed under subsection (d). Based upon credible evidence and testimony presented at trial, the court shall, whether the dog was initially complained of as a nuisance dog

or as a dangerous dog: (i) dismiss the complaint; (ii) deem the dog a nuisance dog; or (iii) deem the dog a dangerous dog. The decision of the court shall be final and conclusive upon the parties.

(g) If a court affirms an order of euthanasia, the owner or keeper of the dog shall reimburse the city or town for all reasonable costs incurred for the housing and care of such dog during its impoundment and throughout the appeals process, if any. Unpaid costs shall be recovered by the municipality in which the owner or keeper of the dog resides on behalf of the hearing authority by any of the following methods: (i) a lien on any property owned by the owner or keeper of the dog; (ii) an additional, earmarked charge to appear on the vehicle excise of the owner or keeper of the dog; or (iii) a direct bill sent to the owner or keeper of the dog.

All funds recovered by a municipality under this subsection shall be transferred to the organization or entity charged with the responsibility of handling dog complaints and impoundment. If the organization or entity falls under the management or direction of the municipality, costs recovered shall be distributed at the discretion of the municipality.

If the court overturns an order of euthanasia, the city or town shall pay all reasonable costs incurred for the housing and care of the dog during any period of impoundment.

(h) If an owner or keeper of a dog is found in violation of an order issued under this section, the dog shall be subject to seizure and impoundment by a law enforcement or animal control officer. If the keeper of the dog is in violation, all reasonable effort shall be made by the seizing authority to notify the owner of the dog of such seizure. Upon receipt of such notice, the owner may file a petition with the hearing authority, within 7 days, for the return of the dog to the owner. The owner or keeper shall be ordered to immediately surrender to the licensing authority the license and tags in the person's possession, if any, and the owner or keeper shall be prohibited from licensing a dog within the

commonwealth for 5 years. A hearing authority that determines that a dog is dangerous or a nuisance or that a dog owner or keeper has violated an order issued under this section shall report such violations to the issuing licensing authority within 30 days.

(i) Orders issued by a hearing authority shall be valid throughout the commonwealth unless overturned under subsection (d) or (f).

Part I ADMINISTRATION OF THE GOVERNMENT

Title XX PUBLIC SAFETY AND GOOD ORDER

Chapter 140 LICENSES

Section 136A DEFINITIONS APPLICABLE TO SECS. 137 TO 174F

Section 136A. The following words as used in sections 137 to 174F, inclusive, shall have the following meanings unless the context requires otherwise:

"Adoption", the delivery of a cat or dog to a person 18 years of age or older for the purpose of taking care of the dog or cat as a pet.

"Animal control officer", an appointed officer authorized to enforce sections 136A to 174F, inclusive.

"Attack", aggressive physical contact initiated by an animal.

"Commercial boarding or training kennel", an establishment used for boarding, holding, day care, overnight stays or training of animals that are not the property of the owner of the establishment, at which such services are rendered in exchange for consideration and in the absence of the owner of any such animal; provided, however, that "commercial boarding or training kennel" shall not include an animal shelter or animal control facility, a pet shop licensed under section 39A of chapter 129, a grooming facility operated solely for the purpose of grooming and not for overnight boarding or an individual who temporarily, and not in the normal course of business, boards or cares for animals owned by others.

"Commercial breeder kennel", an establishment, other than a personal kennel, engaged in the business of breeding animals for sale or exchange to wholesalers, brokers or pet shops in return for consideration.

"Commissioner", the commissioner of agricultural resources.

"Dangerous dog", a dog that either: (i) without justification, attacks a person or domestic animal causing physical injury or death; or (ii) behaves in a manner that a reasonable person would believe poses an unjustified imminent threat of physical injury or death to a person or to a domestic or owned animal.

"Department", the department of agricultural resources.

"Domestic animal", an animal designated as domestic by regulations promulgated by the department of fish and game.

"Domestic charitable corporation kennel", a facility operated, owned or maintained by a domestic charitable corporation registered with the department or an animal welfare society or other nonprofit organization incorporated for the purpose of providing for and promoting the welfare, protection and humane treatment of animals, including a veterinary hospital or clinic operated by a licensed veterinarian, which operates consistent with such purposes while providing veterinary treatment and care.

"Euthanize", to take the life of an animal by the administration of barbiturates in a manner deemed acceptable by the American Veterinary Medical Association Guidelines on Euthanasia.

"Hearing authority", the selectmen of a town, mayor of a city, the officer in charge of the animal commission, the chief or commissioner of a police department, the chief or commissioner's designee or the person charged with the responsibility of handling dog complaints in a town or city.

"Keeper", a person, business, corporation, entity or society, other than the owner, having possession of a dog.

"Kennel", a pack or collection of dogs on a single premise, including a commercial boarding or training kennel, commercial breeder kennel, domestic charitable corporation kennel, personal kennel or veterinary kennel.

"License period", the period of time for which a municipal licensing authority prescribes the validity of a dog license, including the date of issuance of the license through the date on which the license expires, inclusive.

"Licensing authority", the police commissioner of the city of Boston and the clerk of any other municipality.

"Livestock or fowl", a fowl or other animal kept or propagated by the owner for food or as a means of livelihood, deer, elk, cottontail rabbit, northern hare, pheasant, quail, partridge and other birds and quadrupeds determined by the department of fisheries, wildlife and environmental law enforcement to be wild and kept by, or under a permit from, the department in proper houses or suitable enclosed yards; provided, however, that "livestock or fowl" shall not include a dog, cat or other pet.

"Nuisance dog", a dog that: (i) by excessive barking or other disturbance, is a source of annoyance to a sick person residing in the vicinity; or (ii) by excessive barking, causing damage or other interference, a reasonable person would find such behavior disruptive to one's quiet and peaceful enjoyment; or (iii) has threatened or attacked livestock, a domestic animal or a person, but such threat or attack was not a grossly disproportionate reaction under all the circumstances.

"Personal kennel", a pack or collection of more than 4 dogs, 3 months old or older, owned or kept under single ownership, for private personal use; provided, however, that breeding of personally owned dogs may take place for the purpose of improving, exhibiting or showing the breed or for use in legal sporting activity or for other personal reasons; provided further, that selling, trading, bartering or distributing such breeding from a personal kennel shall be to other breeders or individuals by private sale only and not to wholesalers,

brokers or pet shops; provided further, that a personal kennel shall not sell, trade, barter or distribute a dog not bred from its personally-owned dog; and provided further, that dogs temporarily housed at a personal kennel, in conjunction with an animal shelter or rescue registered with the department, may be sold, traded, bartered or distributed if the transfer is not for profit.

"Research institution", an institution operated by the United States, the commonwealth or a political subdivision thereof, a school or college of medicine, public health, dentistry, pharmacy, veterinary medicine or agriculture, a medical diagnostic laboratory, a biomedical corporation, or biological laboratory or a hospital or other educational or scientific establishment within the commonwealth above the rank of secondary school which, in connection with any of the activities thereof, investigates or provides instruction relative to the structure or function of living organisms or to the cause, prevention, control or cure of diseases or abnormal conditions of human beings or animals.

"Shelter", a public animal control facility or other facility which is operated by an organization or individual for the purpose of protecting animals from cruelty, neglect or abuse.

"Veterinary kennel", a veterinary hospital or clinic that boards dogs for reasons in addition to medical treatment or care; provided, however, that "veterinary kennel" shall not include a hospital or clinic used solely to house dogs that have undergone veterinary treatment or observation or will do so only for the period of time necessary to accomplish that veterinary care.